A Oh, other people did studies and said, well, we did not find that, so it must be wrong.
Q So there was a debate in the scientific literature about whether Falck was correct?
A There was some disagreement about it, yes. Now, I think now things are beginning to come back with the congener specific analysis. They are going to come back and probably validate what they put forward in '92.
Q What Falck, et al., put forward in '92?
A Yes.
Q But Falck and his co-authors were looking at PCBs generally, not specific congeners?
A That's right. They looked at, as I said, a summation technique.
Q And that was the technique that we saw cited in later literature where the author said it didn't show any association?
A Yes.
Q And what Falck actually does say is on page 145, "The finding of higher tissue levels among cancer cases may also signify a redistribution of chemicals to the breast during the disease process." Do you see that?
A Yes.

Q Do you agree with that statement?
A I think subsequent events would indicate that is probably not occurring.
Q That subsequent examination of specific levels of specific congeners --
A Yes, where they looked at blood fat PCBs and -- you know, this was a tissue issue. There is no reason to think that patients with breast cancer would have higher blood PCB levels.
I mean, there is just no precedent for that. In fact, there is not even any data to support the notion that there is distribution of greater number of PCBs into the breast tissue of a patient with breast cancer. I mean, there is no biological support for that notion.
Q But why do scientists look at breast tissue and calculate PCB levels in breast cancer patients as opposed to looking at -- I don't know -- legs or toes?
A Because breast tissue is a fatty tissue and the chemical accumulates in fat.
Q Is lipid filled?
A Yes. But you are also interested in disease in that organ. You want to know is there a concentration in that organ of this chemical.
Q So Falck, et al, did not calculate relative

risk; is that correct?
A No, they didn't. All they did is 20 patients and 20 controls. It is not a population study. You can't do relative risk. What you are doing here is a biomarker study.
Q Simply showing a correlation between a level of --
A Right. As they say in the brief introduction, this class of compounds is a good candidate for being a risk factor for breast cancer. That is why they looked at it.
Q That is really all they are doing, to try to find out if it is a risk factor?
A Right.
Q Not to find out to what extent its risk factor or what dose level --
A No, there is no quantification intended or implied here.
Q And --
A And what it does simply say is that the higher the exposure, presumably the higher the risk.
Q Does it say that?
A No, I said it implies. That is the implication of the study and that is why it created such a stir, because it raised the possibility, oh, my God, there may

be this chemical that is in every single person in the United States.
It is in half the foods that we eat and it causes breast cancer, maybe that is why the rate of breast cancer has doubled in last 20 years.
Q PCBs are in half the food we eat?
A Oh, yeah, just like dioxins. Particularly in farmed salmon.
Q Right. We talked about that.
A But there is a lot of other foods where it is present not in such high amounts like in salmon, but it is present.
THE WITNESS: Time for a break?
MR. HOPP: Let's take one.
(Brief recess.)
(Defendants' Exhibits 137 was marked for identification by the court reporter.)
BY MR. HOPP:
Q Handing you what we have marked as Exhibit 137. This is the Hansen article referenced in your report at number seven under breast cancer; correct?
A Yes.
Q This actually talks about male breast cancer after occupational exposure to gasoline and vehicular combustion products; right?

A Yes.
Q What relevance does it have to Sherrie Barnes?
A Well, that's a good question. The mechanism of breast cancer in men is possibly different than the breast cancer in women.
I mean, men and women's breast cancer may have a different etiology. I think probably the important issue here is this would be support of these chemicals that were, in this case, particularly the benzene and the PAHs are present in our case here.
And the implication of the study was that there was an increased risk that they thought was attributable to these exposures and this is a one case report.
It is not terribly important to our overall case, but it is -- let's go to the last paragraph where he discusses this issue.
He basically talks about, "The
Elevated risk of breast cancer
Among men, occupational exposed
Gasoline and combustion products
Has not been reported previously
Except in one small study with
nonsignificant odds ratio of 1.3.
However, two recent studies show an
increase in breast cancer in women

exposed to benzene and PAHs."
Which is the Petralia study, I believe is also on this list. I know it is on my new list.
And it is here on this list, and then he states, "The similarities among some of
The known risk factors for breast
Cancer in men and women and a
Similar variation in incidents
Point to common etiologic factors;
therefore, gasoline and combustion
products caused breast cancer in
Men. It probably does so in women,
too."
And then it goes on to discuss some other things.
Q So the author is hypothesizing that this result that he obtained in this paper might be applicable to women, as well; is that fair?
A Yes, and then he alludes to some other studies that showed he doesn't do an exhaustive review. Where we actually know that there are other papers that he could have cited.
Q Sure. And we will get to those.
A But the point is that it is just another study of a case of someone who has some pretty good exposures

to these chemicals that developed a rare disease.
Oftentimes rare diseases, like men's breast cancer or mesothelioma can give us a lot of clues and should be followed up when they occur.
Q Would it be fair to characterize the Hansen paper as generally informative, but not directly related to the cause of Sherrie Barnes' breast cancer?
A Yes.
Q In fact, the article does not calculate the relative risk for breast cancer in women; is that right?
A Correct.
Q And at what exposure level does the study indicate that breast cancer has increased in men?
A Well, he has got an odds ratio here of 2.2 with no lag time and 2.5 with ten years of lag time with statistical significance.
Q Lag time being years of exposure? What does lag time mean?
A No. What that does is it allows for more latency.
Q Okay.
A In other words, you look at the people's exposure and then you make sure that you are at least allowing for ten years of lag time from the time of exposure to the time of the disease diagnosis.

Q Is it accurate to say that the Hansen study doesn't examine specific exposure levels, but rather looks at occupational exposure of gasoline and combustible products in general?
A Yeah, 230 male employees were members of the National Pension Fund and the country is Denmark. And he looks at job title for exposure.
Q Okay. So there is no exposure data for the individual study subject?
A No.
Q Next one on your list -- your breast cancer reference list number eight is the Holford, H-O-L-F-O-R-D, study?
A Yes.
Q Handing you what we have marked as Deposition Exhibit No. 138. This is a copy of the Holford study. The Holford study is entitled Joint Effects of Nine Polychlorinated Biphenyl (PCB) Congeners on Breast Cancer Risk; is that right?
(Defendants' Exhibits 138 was marked for identification by the court reporter.)
THE WITNESS: Yes.
BY MR. HOPP:
Q And Holford looked at nine PCB congeners; right?

A Yes.
Q And, generally, what did Holford conclude?
A There is an association with some of them. Let's see if I can make sense out of this.
Table 2 shows odds relative risk associated with a ten PB change in exposure to individual congeners by type of model; and I think the risk associated congener values that are listed in the middle .2153 and 156 is not being significant.
Q Okay.
A But 183 is significant.
Q 180 is slightly elevated, but not significant; right?
A Yes, 180 is slightly elevated, but it is not very significant. It is almost significant, it is .99. It is real close. Anyway --
Q But 183 is the culprit in that Holford paper; right?
A That is the one that they felt was statistically significantly associated.
Now, on Table 3, they give an odds ratio associated with a level of PCB in quintiles and they divided them into five levels.
Q I'm sorry. Table 3?
A Table 3, it is at the bottom of 979.

Q Okay.
A And there, they -- as the level of the PCB increased, the odds ratio of relative risk -- I think it is related risk score, which is similar to relative risk, it is adjusted estimates of relative risk. Risk becomes statistically significant only at the top quintile. Otherwise, the curve is pretty flat.
Q And Table 3 is looking at all of the congeners that are being studied or is it --
A They have some kind of PCB score. It is a score -- let's see how they scored it. Somewhere in here they describe the score.
All right. Well, it is on Page 977. It is called Principal Components, and they describe what they did.
"In order to understand better
the nature of the effects for
individual congeners, principal
components analysis was used
to create factors that were
independent of each other.
Using PRO PRINCOMP in SAS we
estimated the eigenvectors, which
provided loading scores that gave
rise to new variables to be

included in linear logistic model."
Q What the heck does that mean? Do you understand that?
A Yeah, they are doing statistical analysis, which is -- when you have multiple variables like this, you know, a dozen or so PCBs, plus other variables, age and whatever else you put in the model, you have got a very complex statistics; but not being a statistician, I cannot really explain to you what they are doing. It is a very high order statistical.
Q Well, principal component analysis is the general name for what they did?
A Yes. Well, that component.
Q All right.
A In the Statistical Methods, they discuss their analysis, how they did it, and one they want to look at the joint effects of individual PCB congeners on the risk of breast cancer and whether the effect of each congener was the same, which was tested using linear contrast.
"If these results suggested
That the magnitude of effect on
Breast cancer risk was different
From the congeners, then it
Would not make sense to evaluate

Total PCB exposure, but to
Investigate the joint effects of each
congener. Regression diagnostics
Were used to determine whether
The results were sensitive to one or
more influential observations."
Q I'm sorry.
A Now, we are talking about regression diagnostics was used on one or more influential observation.
"But the overall conclusions
Were found to be stable. Bootstrap
methods were used to estimate
Bias in the estimates of risk, as well
as providing alternative estimates of
standard errors. While the resulting
standard errors were slightly greater,
the conclusions were essentially
unchanged, so these results are not
present."
I think what they are saying is that their principal component analysis is what they used and that is what they used in Table 3 as a related risk score.
Q And above Table 2, the authors point out, the statement is, "Notice that some congeners

Are positively associated with breast cancer risk, while others are negative"; is that right?

A Well, if you look at the standard coefficient, the first line, when it says, negative .021, that means that the higher the PCB level of the congener, the lower the breast cancer risk.

Q All right.

A So that is right. There were three -- four that were negative and then one, two, three, four, five that were positive.

And 180 was the most positive statistical and it reached almost statistical significance and 183 did.

Q And in the Discussion section, this is on Page 979, the authors point out that, "The Association of total PCB exposure with breast cancer risk in this analysis was estimated to be small and inverted." Is that what you are talking about?

A Yes, for those who had it -- the higher the level, the lower the risk, suggesting -- I think, you know, you can find and do these fancy statistics. You can find things like this. That may not mean anything.

The most important thing here is to look at all the congener correlations, and 180 and 183, again,

correlates strongly with the total congeners.

In other words, you are getting a positive effect on the breast cancer. And like the other studies we have looked at, if you add up all of the PCB congeners, that also correlates with breast cancer risk.

So what it would suggest is that the overall mixture, maybe some components being more important than others, is contributing to the risk; and that the negative components do not outweigh the positive components in terms of causing the effect that we are seeing in the increased risk.

Q But they do balance out and that is why the authors say that the overall risk is small?

A That's correct.

Q And they go on in the Discussion section and say, "These results suggest that some Congeners have a protective effect on breast cancer risk, while others are associated with an increased risk"; is that right?

A That's right. That is correct.

And I think that is consistent with all the data. It shows that there is a small but significant increase in risk. And the reason it is important is that there are so many darn people exposed and so many

people get this disease, that anything that contributes to the risk is important to address.

Q Is this a case control study?

A This is a biomarker study. I mean, there is cases and controls. What they are doing is they are studying the presence of a biomarker in PCBs in two populations to see if the testing hypothesis that the cases would have a higher level of these chemicals than the controls.

And the answer is, yes, and it does show correlation.

Q Does this study indicate what dose of any particular PCB congener is necessary to cause an increased risk of breast cancer?

A No, I mean, if you look at the -- I don't think there is a single measurement in this whole paper. It is all statistical analysis.

Let me just see. Maybe they are mentioned somewhere. The level -- no, what they are really trying to do is the correlation or the association of the chemical versus the risk. And that is not going to give you thresholds or slope factors.

Q The next paper in order on your reference list, this is number nine, is the Hoyer paper; is that right?

A Yes.

Q I am handing you what we have marked as deposition Exhibit No. 139.

(Defendants' Exhibits 139 was marked for identification by the court reporter.)

BY MR. HOPP:

Q Is this the Hoyer paper?

A Yes.

Q And it is entitled Organochlorine Exposure and Risk of Breast Cancer. What question was Hoyer trying to answer?

A The same question. He looked at Dieldren, which is an organochlorine. He looked at chlorocyclohexane, which is another pesticide, organochlorine pesticide.

Q Did this study look particularly at TCDD or dioxin?

A No, it looked at PCBs, DDE, but it did not look at dioxin per se.

Q So this would be another study that is generally informative, but it is not directly related to Sherrie Barnes; is that right?

A Yes. For the reasons that I indicated earlier, I thought it was relevant.

Q And they actually looked at serum levels; is that correct?

A Yes.
Q So these are blood samples and not tissue samples?
A Yes, serum sampling. That is right.
Q The Result section indicates that,
"The risk of breast cancer decreased with increasing number of full-term pregnancies and increased with" -- I'm sorry -- "and increasing with body weight and height."
Do you see that?
A Where are you reading from?
Q The Result section, this is Page 1818 starting right above that table.
A "Increasing number of full-term Pregnancies and increasing with Body weight and height."
So height was made a standard.
Q You wouldn't think so. But Hoyer at least concludes that increasing body weight and height are a risk factor; is that right?
A This is the first time I have ever seen height as a risk factor for anything. And unmarried women had an 89 percent higher risk than married women. It is probably because they didn't have babies.

influence on the risk of breast cancer."
Is that right?
A Yes, in this study, they did not find an increase in breast cancer. That's correct.
Q The next study in order under List of Breast Cancer References is the -- maybe you can pronounce it for me. Kogevinas paper?
A Kogevinas is as good as any.
Q Kogevinas, K-O-G-E-V-I-N-A-S.
I am handing you what we have marked as deposition Exhibit No. 140, which is the Kogevinas paper.
(Defendants' Exhibits 140 was marked for identification by the court reporter.)
BY MR. HOPP:
Q Now, this is a review article; is that right?
A It is.
Q So it doesn't report on a new experiment, but rather discusses studies done by other people?
A Yes.
Q And does Kogevinas find -- well, let me -- what does Kogevinas conclude, generally, based on the other studies?
A More studies are needed. That was his main conclusion, but he reviews some of the studies and it is

Q Moving on down this page, this is 1818. It says, "We found a slight increase in
Risk of breast cancer with increasing concentrations of BHCH, but no association was apparent for total DDT or total PCBs."
Do you see that?
A Um-hmm.
Q So this study tends to conflict with some of the other studies which have indicated PCBs increase the risk of breast cancer?
A Well, they did 28 PCBs. They don't tell us which ones. So this wasn't as detailed a congener analysis as the others.
They do list them here. And -- yes, they just didn't find a correlation.
Q And then the Conclusion, which is on the last page states, "Our results support the
Hypothesis that organochlorine compounds, such as dieldrin,
Which have oestrogenic properties,
May increase the risk of breast cancer.
They do not, however, suggest that exposure to total PCB, total DDT,"
And I guess, "P prime-DDE have any

interesting in that respect.
Q And that is, again, generally informative, but not particularly relevant to Sherrie Barnes?
A Correct. He gives a list of the various studies and notes, you know, the breast cancer, including male breast cancer, has been found to be increased.
Q He finds ~~increasing mortality~~ from breast cancer that is not statistically significant; is that right?
A Yes.
Q That is in Table 5?
A Yes. Table 5 he is looking at -- where is that? He has got different references 170 -- where is it? I am trying to see what his references are for that.
Anyway, he -- I guess, IARC's international cohort study of phenoxy herbicides or chlorophenols where TCDD was presumed to be present and the SMRs are elevated for all of the cancers, but all malignant neoplasms are statistically significantly increased.
And the individual types of cancer, breast female is almost statistically significant. The odds of SMR is 2.16, but the confidence interval is at .99. We are talking about 100ths off. Otherwise, it would be

statistically significant.
So that, in light of all the other evidence we have, this is supportive.
Q All right. But you are looking in this paper, Table 5, you are looking at nine deaths; is that right?
A Yes.
Q Out of how many expected?
A Well, that would be 2.16 more than expected. So you would expect in that population -- I guess, the 174 reflects the number of something rather -- what is it? I don't know the number of people at risk; but they expected half of that many cases. So there is a doubling of risk.
Q If the spread at the 95 percent confidence interval includes one, then it is not statistically significant?
A Yeah, I know. And if it was one more, it would be.
That is the point I am trying to make is it is very close to statistically significant; but if the numbers were bigger, it would be significantly.
And as I say, by itself, it would not be important, but taken in light of all of the other evidence, it is supportive.
Now, the same is true of male breast cancer.

It has doubled 2 1/2 times the -- twice as they expected.
And, again, that goes along with our other observations about this and, similarly, prostrate is elevated. Testes is elevated. Thyroid is elevated, and all endocrine organs are elevated. The numbers are small.
Q And not statistically significant?
A Not statistically significant, but the point is all of these cancers are endocrine disruption sensitive cancers. And, again, in view of other information, it certainly is worth paying attention to.
Now, if you go over to the last one,
"All workers exposed to any phenoxy herbicide or chlorophenyl."
Q Still on Table 5; right?
A Still on Table 5. You have got a statistically significant excess of, again, all malignant neoplasms and other endocrine organ cancers are elevated statistically significant.
So it would appear to me that, you know, this paper is useful.
Q In a general way?
A Correct.
Q It does not identify a particular dose level

which is required to increase the risk of breast cancer; is that correct?
A No.
Q The next paper in order on your list of breast cancer references is the Laden paper, L-A-D-E-N; is that right?
A Yes.
Q And it is number 11; correct?
A Yes.
Q I am handing you what I have marked as Deposition Exhibit No. 141.
(Defendants' Exhibits 141 was marked for identification by the court reporter.)
BY MR. HOPP:
Q This is the Laden paper; is that right?
A Yes.
Q And the Laden paper looks at the Nurses' Health Study; is that right?
A Yes.
Q Is that otherwise sometimes called the Harvard Nurses' Study?
A Well, this is from Harvard. So it could be considered the Harvard Nurses' Study.
Q Have you heard that expression before, the Harvard Nurses' Study?

A No. I heard the Harvard Doctors' Study, but yesterday when you mentioned the Harvard Nurses, this is the first I heard of it.
But as I said, this is a study of nurses conducted by Harvard. So it would be appropriately called that.
Q And correct me if I am wrong, but it appears that what happened was Harvard or some group at Harvard has collected and has continued to collect data on a large group of nurses.
It is sort of a prospective study. It examines health effects over the course of the lives of these women?
A Yes, just like the doctors' study. Same idea.
Q The idea is to --
A Follow the large group and see what happens to them and look at the different risk factors prospectively.
Q It states, at the end of the abstract,
"The majority of studies have concluded the exposure to PCB are unlikely to be a major risk factor for breast cancer."
Is that right?
A Are you talking about --
Q I am looking at the end of the abstract.

A Although there is no independent association, blah, blah, blah -- yeah, the point of this paper is that if you look at the nurses who have this particular polymorphism, CYP1A1-exon 7, this is a risk factor for breast cancer.
Q Okay.
A And I think what they found was --
Q Was what he found that this was a genetically susceptible population?
A Correct.
Q Okay. Doctor, do you want to continue with your answer?
A What they say here is, "However
High levels of PCBs may be associated with breast cancer risk in the subgroup of women who have variant CYP1A1-exon 7 polymorphism."
Additional studies are needed to examine that possibility.
Q That is CYP1A1-exon 7 polymorphism, that is something to do with the particular genetic structure of these women; is that right?
A Yes.
Q It is a gene?
A Their ability to transform the PCBs or handle

or not it would be relevant to test her.
Q This study looks at latent PCBs; correct?
A Correct.
Q Doctor, I have handed you what we have marked as Exhibit 142. This is the next reference on your breast cancer list. It is number 12 and the author is Leis or Lees. L-E-I-S.
(Defendants' Exhibits 142 was marked for identification by the court reporter.)
THE WITNESS: Yeah.
BY MR. HOPP:
Q And this is really just a paper on diagnosing breast cancer; is that right?
A Yes, it has risk factors. That is the reason it is here.
Q But does it talk about environmental risk factors or TCDD?
A Not really, it talks -- Table 1 and Table 2, exogenous estrogen, which would be in birth control pills and hormone replacement. And then it says,
"Carcinogenic exposure, particularly to viral agents and some drugs."
Q So --
A Really, it just kind of gives you a list of

them is impaired or reduced.
Q And that's the only study -- strike that.
That is the only population in which the Laden paper found an effect with increased levels of PCB; is that right?
A That's right.
Q And we don't know whether Sherrie Barnes had that particular polymorphism, do we?
A No. You asked me that yesterday. So we don't have any studies on Sherrie Barnes or anybody else on cohort. It is not a routine thing you send to the lab.
Q You have to take a tissue sample?
A You have to do genetic studies. That is what you have to do to find this particular variant. It is expensive and it is possible to be done. But it is very important particularly in people that we don't have disease in yet; but we want to know who is at high risk. These kind of studies would be highly relevant.
Q Would it be possible to test Kenesha Barnes to find out whether her mother had that particular polymorphism?
A Well, we would have to check her dad, too. I don't know how the inheritance goes for that particular gene. I don't know if it can be an acquired defect. I would have to study it to answer that question whether

things that have been raised as -- just kind of a general review of the disease. So you know what you are talking about.
Q Not very informative with respect to causation?
A Correct. I don't think he has references for a lot of those causative factors. He doesn't give a reference. He makes the assertion in this table.
Q The next breast cancer reference that you have in order, number 13, Lucena, L-U-C-E-N-A; is that right?
A Right.
Q I am handing you what we have marked as Exhibit 143.
(Defendants' Exhibits 143 was marked for identification by the court reporter.)
BY MR. HOPP:
Q This is the Lucena paper; is that right?
A Yes.
Q It is entitled Short Communication. Is this -- is there some significance to that?
A Well, what they do is they write a very brief paper presenting one table, maybe, which they think is important when they want to publish it as a -- quickly, so it is easier for the reviewers to deal with a short paper with very little information, so you can get it published faster.

Q Right. And this paper really identifies one specific congener or PCB?
A 28.
Q As associated with an increase risk of cancer; is that right?
A Yes. Fascinating. It is like every paper has a different congener. Congener of the week.
Q Did Lucena look at other congeners?
A They looked at a bunch of them. It is listed on the top of 118, left-hand column.
Q But the only one they found that significantly increased the risk of breast cancer was 28; correct?
A Yes.
Q Once again, they think there is a great need for more studies?
A That's, as I told you, every study will say that. It is the stock and trade of a researcher.
Q Does Lucena calculate a relative risk for exposure to PCB 28?
A Yes, 9.597, huge odds ratio. Same thing.
Q But it does not identify a particular dose level for that congener which would result in that increase risk; is that correct?
A I don't see that it was quantified. What they said was in the difference between the exposed and the

controls, it was a ninefold difference in that chemical.
Q So what they were – this was a study in Spain; is that right?
A Yes.
Q And they were actually looking at breast tissue that had been removed from women who had breast cancer; correct?
A Yes, that's correct.
Q And these were malignant lesions?
A Well, in the exposed, they were malignant.
Q And the controls, they were benign lesions; is that right?
A Benign lesions.
Q So what they found was that if someone had a detectible level of PCB 28 in the malignant lesion, those people turned out to have a 9.597 odds ratio; is that correct?
A That's right.
Q How is this paper – strike that.
How does this paper relate to or inform your opinion with respect to Sherrie Barnes?
A The same as the other PCB papers. We are talking about a similar toxicity for dioxin-like chemicals.
Q The next one in order on your breast cancer

reference list is the Manz paper; is that right, M-A-N-Z.
A Yes.
Q I have marked that deposition as 144. This is the Manz paper; is that correct?
(Defendants' Exhibits 144 was marked for identification by the court reporter.)
THE WITNESS: Manz paper, correct.
BY MR. HOPP:
Q M-A-N-Z?
A M-A-N-Z, from Germany.
Q This is a German study of exposure, actually, of workers in a chemical plant; is that right?
A That's correct.
Q And they characterized – first of all, it is a retrospective mortality study; correct?
A Yes.
Q And they characterized the herbicide workers in this plant in Germany as being having been exposed to heavy contamination of 2, 3, 7, 8-TCDD?
A Yes.
Q But only seven percent of the women worked in high exposure areas of the plant; is that right?
A Yes.
Q Did they detect an increased risk of breast

cancer as a resulted of heavy exposure of 2, 3, 7, 8-TCDD?
A I think this is overall, the SMR for carcinoma of the breast was 2.15 with a 95 percent confidence interval of 0.98.
Again, right at the borderline, and 409 for nine deaths.
Q And this is what table?
A It is on Page 962 under Mortality Among Women.
Q All right.
A Malignant neoplasms were right at not significant, but the breast cancer was. And that's really the point of the paper, which is about TCDD.
Q Okay. So it is just about TCDD, and the 2.15 is an increased SMR, but is it statistically significant?
A Well, it is right at that borderline at 0.98.
Q Again, the 95 percent confidence level includes one?
A That's right. It is right at the borderline.
Again, I think I have said it before, when it is taken into the context of everything else, it is supportive. They also review a study, which I don't think we got, but –
Q Which study is that?

A I am just looking at it here. I am wrong. That's the only point of this study.
Q Does the Manz paper identify a dose level of 2, 3, 7, 8-TCDD, which is significant for increasing the risk of breast cancer?
A No.
Q Is the exposure level documented in the Manz paper?
A No, they don't do blood levels or the chemical plant was found to have high TCDD levels enough to cause chloracne. And that was led to the change in practices to reduce exposures.
Q So qualitatively, they think it was high because of the chloracne?
A Well, we know that when you get chloracne, you are at high levels; but they don't give the numbers in here.
Q While we are on the subject of chloracne, I know I discussed this with Dr. Sawyer, and forgive me if I covered this with you.
Are you familiar with the case of Victor Yushchenko?
A Yes, I am.
Q Victor Yushchenko is the president of the Ukraine; is that right?

A Yes, he is.
Q He was actually -- someone tried to poison him with dioxin?
A That's right.
Q Do we know if it was 2, 3, 7, 8-TCDD?
A No, we don't know precisely, but he had dioxin poisoning. And in the poisoning episode, they usually use TCDD because it is available. If you are running a lab when you are testing this, you can get TCDD as a standard.
Q You could have gone to the German factory and seen the Manz paper and gotten it?
A Yeah, I guess so. You can get purified TCDD from a chemical supply house.
Q Now, the acute exposure to -- strike that.
I believe Dr. Sawyer testified that the level of Victor Yushchenko's exposure to TCDD was among the highest ever recorded?
A Among the highest recorded, that is correct.
Q There were a couple of other acute poisoning cases that were documented, several women 20 years ago or so, who were up in that range, as well; is that right?
A Yes, from Vienna, Austria.
Q But Victor Yushchenko did not die from his

poisoning; correct?
A Not yet.
Q How about the women in Vienna, Austria; did they --
A They have not died yet either, but they are being followed. They are about ten years from the onset of exposure. And --
Q Did they --
A They are quite ill and I suspect Victor Yushchenko is quite ill. They have been attempting to get the levels down using various techniques to detoxify, but nothing is working. But the levels of both the two women from Austria and Yushchenko are still extremely high.
Q They were using Olestra, I think, with Yushchenko; is that right?
A They used Olestra with the two ladies from Vienna, also.
Q Did it work?
A It is a miserable, miserable drug. It causes diarrhea and people can't take it. So they take it for a while till they get sick of it. It may lower the level a bit. It is not terribly effective.
Q Olestra is the fake fat; right?
A That's right. The non-absorbable fat. It is

the same as Cholestyramine and the cold pressed oils that we use. It compresses (phonetic) in the gut.
Q And the women from Vienna, have they developed breast cancer?
A No, not yet. And we talked about this earlier, it may not be TCDD in the adult that causes the breast cancer, anyhow. Or it may not be nearly as potent a factor in the equation. I mean, you can induce --
Q Let's move on. The next paper you have cited in your breast cancer references, it is number 15, the Morris paper; is that correct?
A That's right.
Q I am handing you what I have marked as Deposition Exhibit No. 145. This is the Morris paper; correct?
(Defendants' Exhibits 145 was marked for identification by the court reporter.)
THE WITNESS: Yes.
BY MR. HOPP:
Q And this paper is -- would it be accurate to call the Morris paper a hypothesis-generating paper?
A Well, he reviews all the data. That's the value of reading a paper like this.
Q What, if anything, does Morris conclude?
A Well, he talks about benzene, benzopyrene. He

does talk about cigarettes, aromatic hydrocarbons, and breast cancer, and PAHs.
Q Is Morris a review paper?
A Yes. He goes on to talk about PAHs and in quite a bit of detail. And then concludes, you know, that something going on in our environment is causing this. And his candidate is aromatic hydrocarbons, in a broad sense.
And he reviews a bunch of them. And, of course, PAH is at the top of the list here. He does not go into much detail on the polychlorinated hydrocarbons.
He is mainly focused on the aromatic hydrocarbons. It is a very thorough review of those papers up to that time.
Q It is sort of a dated paper; right, this is '92?
A '92, but there was still quite a bit more evidence already at that time.
Q Morris identifies radiation and aromatic hydrocarbons as inducing and promoting mammary cancer; is that correct?
A Yes, that's correct.
Q He also states that such disparate factors as urban residents, geographic location of residents, and life-style factors, such as alcohol ingestion, high

polyunsaturated fat diet, and food selection and preparation all contribute to exposure to promoter and initiating influence of aromatic hydrocarbon carcinogenesis; is that right?
A That's correct. That is what he says.
Q Does Morris isolate any particular exposure, any particular PAH which he thinks is significant for causing breast cancer?
A Benzopyrene and DB(AH)A anthracene, which are the experimental animal carcinogens. He also mentioned DMBA and PAHs in general.
Q In his review, does he discuss human epidemiology studies, or any the animal studies and in vitro studies?
A Well, he does -- he touches on animal studies quite a bit. Because in '92, there were fewer studies, but he mentions benzene, as well, and its ability to induce cancer, and talks about the -- mostly the animal study.
There wasn't as many studies back at that time in humans as there are now. But he gives a background as to why people started looking so hard at human studies, subsequently.
And he points out why these chemical PAHs, in particular, are likely to be the cause of breast cancer.

Q He does not address creosote as a mixture; correct?
A No, he doesn't.
Q And does he document any exposure levels to any particular PAHs?
A No.
Q Does he calculate relative risk levels?
A No, he doesn't do that either. This is a review paper of pointing out all of the papers that exist at that time that point towards a link between the PAHs and breast cancer.
Q I understand, but Morris does not identify any particular exposure level that is necessary to produce harm; correct?
A No.
Q I'm sorry. That was a bad question.
Does Morris identify a particular exposure level that is necessary to produce harm?
A No, he doesn't.
Q The next paper on your list of breast cancer references is number 16, Muscat, M-U-S-C-A-T; correct?
A Yes.
Q I am handing you what we have marked as Deposition No. 146.
(Defendants' Exhibits 146 was marked for

identification by the court reporter.)
BY MR. HOPP:
Q This is the Muscat paper; correct?
A Yes, it is.
Q Entitled Adipose Concentrations of Organochlorine Compounds and Breast Cancer Recurrence in Long Island, New York; right?
A Yes.
Q So, again, he is looking at PCBs; right?
A Yes.
Q And what, if anything, does Morris conclude?
A Muscat.
Q I'm sorry. Muscat conclude?
A That there is a linkage between adipose PCB levels, which is -- let me see. I think it is recurrence in -- of breast cancer.
Q Muscat is looking at cancer coming back a second time?
A Yes, he is talking about it being a predictor of recurrence of breast cancer. Interesting study.
Q How does this relate to Sherrie Barnes?
A Again, it is showing PCBs which are dioxin-like in their behavior increasing the risk of recurrent cancer, which is relevant to our patient, I believe, in the sense that she had a tumor that was very aggressive.

What they are suggesting here is that PCBs probably increased breast cancer risk, but that is not the main point. The main point is that it is -- or associated with recurrence.

Q So Sherrie Barnes had it once and it was fatal?

A Yes. She did not have a recurrence. She did not respond to the therapy either, suggesting that her tumor was very aggressive and malignant.

And this paper suggests that making the tumor grow more readily would be associated with these types of exposures.

Q And in the concluding paragraph, they point out that these results, that is, the results represented in deposition Exhibit 146, conflict -- I'm sorry -- contrast with the author's previous data showing no effect of organochlorine compounds on breast cancer in these women; is that right?

A Yes.

Q So there was a previous paper by the same authors which was negative; correct?

A That's right.

Q Does the Muscat paper calculate relative risk of recurrence?

A Let's see, relative risk is on Table 5, and as the level grows, most of the relative risk grows

patients all the way -- there are three or four that aren't.

And no one stands out, but then the total turns out to be statistically significant. And the biggest difference is in the blacks. Where they -- it is 129 parts per billion difference.

Q Can you explain that? What does it say about black women?

A That blacks with no recurrence, their PCB total was 406. The blacks with recurrence, their PCB level was 529. Both of those values were higher than the whites.

And the highest at all are the Asian with no recurrence, but there is very small number of Asian, so --

Q So does the Muscat paper identify an exposure level as necessary to cause harm?

A No, they do not.

Q Muscat does indicate on, Page 1477, that there were relatively few events in this study and the positive findings could have been due to bias?

A Sure.

Q What is bias in this context?

A Something that is causing the results that is not a true cause. Bias just means that there is

significantly with each PCB congener.

It is interesting. Not all of them were that way, but starting with -- with the 74, lowest tertile was 1; middle tertile was 1.3; and highest tertile 1.7.

And then, anyway, they go all the way down. Some of them are statistically significant. Some aren't. The total PCBs is most significant at the highest tertile. 2.9 is the relative risk with the statistical significance.

Q This paper actually does contrast with some of the other papers we looked at, even today, which show that some of these same congeners do not increase the risk of breast cancer; correct?

A Yeah, I think it would be -- they need bigger numbers, probably, to do that, but more importantly --

Q Explain that. Who would need bigger numbers to do what?

A Well, how many patients did they have? 30 patients in the recurrence category.

Q You are talking about Muscat?

A Muscat. If you had maybe 300, you might be able to start seeing differences in the individual congeners; but they do have mean concentrations in the blood of the various congeners and consistently -- pretty consistently, they are higher in the recurrence

something that is screwing it up.

Epidemiology people always say those sorts of things. It is just terms of epidemiology.

It could have been through chance. It could have been bias. We don't know. We tried to remove all the bias; but there is always a risk. Something that we didn't control for.

Q Isn't that what epidemiologists spend most of their time doing? Try to eliminate possibility of their chances; influencing their --

A Yes, they spend a lot of time.

Q That is the whole point. If the result is dictated by chance, then you have wasted your time doing your --

A Exactly. You are going to get a negative study. That is why they tighten, over the years, the criteria to say significant.

It used to be, when I started out in medicine, P value of .1 was considered significant. Now, it is .05. So you have to have a really good study, really a strong effect to get statistical significance.

Q Your next study on your list of breast cancer references is the Negri, N-E-G-R-I, study; is that right?

A Yes.

Q Handing you what I have marked as Exhibit 147.
(Defendants' Exhibits 147 was marked for identification by the court reporter.)
BY MR. HOPP:
Q This is the Negri study; right?
A Um-hmm.
Q This is a review article; right?
A It is.
Q And it looks at exposure to PCB and breast cancer?
A Yes.
Q And what does Negri and/or her coauthor conclude?
A Well, I think the important point is that you need to take into account genetic susceptibility in order to explain what is going on; and that in the general population, without the genetic risk factor, there probably isn't an increased risk.
Q So, in fact, at the concluding part of the study, right above the acknowledgments, Negri and coauthors say, "In conclusion, the epidemiological evidence does not support the hypothesis of a direct relation between environmental exposure to PCB

of the related compound.
Q So it is generally informative, but not directly related?
A Correct.
Q The next paper in order on your breast cancer reference list is Petralia; is that right?
A Yes.
Q It is Petralia, 1999?
A Yes.
Q Petralia has written several articles on this subject; right?
A I have Petralia -- another one of the Petralia papers on the new -- that I gave you. Two more, '95 and '98.
Q So you have got some older papers?
A '98 and '99. So I have got the '99 paper, but I have got an earlier '98 paper that I have added.
Q Let me show you Exhibit 148.
(Defendants' Exhibits 148 was marked for identification by the court reporter.)
BY MR. HOPP:
Q This is 1999 Petralia paper?
A Yes.
Q And Petralia is looking at the premenopausal -- I'm sorry, risk of premenopausal breast cancer in

adulthood in the general population and the risk of breast cancer"; right?
A That is what he said in the abstract, which I just read to you.
Q And then he goes and talks about a specific genetic variation like --
A Right. He is really just repeating what we said earlier about the CYP1A1 and the exon 7. He does not mention exon 7, but in Table 5, he mentioned that.
Q But for the general public, Negri is, essentially, a negative paper; right?
A Yes, that's the point. But when you take into account the -- see, there is a couple of papers that we have not gone through that are reviewed here that make the same point.
Interaction between PCB and the CYP1A1 polymorphism, I think what the science has evolved to the point that it takes -- you can have the CYP1A1 gene and not get breast cancer; but if you have it and are exposed to PCBs, then your risk of breast cancer increases significantly.
Q And how is this study relevant to Sherrie Barnes?
A Well, it is like all of the others. I have referred to in the PCB literature. It shows the effect

association with occupational exposure to polycyclic aromatic hydrocarbons and benzene; is that right?
A Yes.
Q So this is an occupational study?
A Yes.
Q And does it look at women particularly in these occupations?
A It has to be.
Q Premenopausal --
A The rate in men, as we know, is quite low. So it is women. And the exposures were variable.
They took occupational history of the exposure assessment for PAHs and benzene was developed to determine which occupations had exposure. And then they developed a matrix for that, which included the PAHs and the benzene and others things, as well.
Q And which exposure levels did they find to be significant to increase the risk of premenopausal in breast cancer in their occupation when exposed to both?
A PAH and benzene, highest risk was in PAH and benzene together. They found statistical significance in all of them and the biggest abnormalities were in the ER positive cases.
Q What is that?
A Estrogen receptor positive, which we looked at

yesterday in our case.
Q Oh, ER positive breast tumors, that is a particular type of tumor?
A Yes, this is the first time we have seen that.
Q Okay. Seen what? Seen a study?
A Seen a study where they looked at the ER positive and ER negative.
Q Forgive me for covering this again, but we don't know whether Ms. Barnes had a ER positive or ER negative breast; right?
A Yes, I don't think we do.
Q Again, forgive me for asking you to say this again, but what was the dose level that the authors of the Petralia paper found to be significant for inducing breast cancer? Did you say it was every dose?
A Well, they have got some duration data here which would be a surrogate for dose.
Q Oh, I see. They use job exposure matrixes and lifetime occupational history; is that right?
A Yes. And they had low exposure and medium to high and then cumulative low, medium to high and, in general, I think they only found a few cases that were a statistically significant; but the numbers in each cell are so small that it is not likely to find statistical significance.

So there were some elevated odd ratios. In fact, lots of them were elevated; but it didn't reach statistical significance, except in a few cases.
And, again, if you look at the ends, that is the problem. There aren't enough in each of the cells to reach statistical significance.
Q So it is too small a study, really, to effectively evaluate statistically significant association?
A With dose. It is a large enough study to say in general. I mean, you have got quite a few people in the exposed categories.
Q So overall, looking at overall exposure, they find an increase risk?
A That's right.
Q But they cannot break that down by exposure classification?
A Right. Of the patients that they looked at, they had 25 of PAH alone; 35 of benzene alone; 6 exclusively PAH; 19 PAH and benzene; and 16 exclusively with benzene. Those are --
Q Small numbers?
A Relatively small numbers, but big enough to get statistical significance on many of these; but then when the numbers drop down to 16, 8, 13, and 11, 10 and 9 --

10 and 8, then they don't get statistical significance even though they have elevated odds ratios.
Q Looking at Page 220, this is the first full paragraph. It says, "When our results are interpreted, several issues need to be considered. The response rates for both the cases and referents" -- that is R-E-F-E-R-E-N-T-S -- "were low."
Now, that is a problem for epidemiology?
A Where are you reading from?
Q Page 220, first full paragraph, starting with the words, "When our results are interpreted."
A Oh, I see, on the right-hand side.
Q Low response rate is a problem for an epi study; right?
A Yes, the response rates were low. That is a problem.
Q And would you characterize it as a case control study or a cohort study?
A Case control.
Q How does this paper, the Petralia paper, relate to Sherrie Barnes?
A She was exposed to both benzene and to PAHs, and so it would be a direct relationship. Although it

was not occupational exposure, she had environmental exposure. I would submit that she probably had higher exposures to PAHs and benzene than the people in this study.
Q Does the Petralia study, then, identify an exposure level that is necessary to cause harm?
A No, it doesn't have any quantitative data.
Q All right. The next study in order on your list of breast cancer references is Pliskova; is that right?
A Yes.
Q P-L-I-S-K-O-V-A. I am handing you what I have marked as deposition Exhibit 149. This is the Pliskova paper; correct?
(Defendants' Exhibits 149 was marked for identification by the court reporter.)
THE WITNESS: Yes.
BY MR. HOPP:
Q I have actually handed you two things. One is the abstract and one is the article.
A Oh, yeah.
Q Let me have the abstract back, so we don't confuse ourselves.
A Okay.
Q So Deposition Exhibit 149 is the Pliskova

article; correct?
A Yes.
Q And Pliskova article states what, specifically?
A Benzopyrene and -- what is the second one? Benzanthracene.
Q Is this an in vitro study?
A This is an in vitro study.
Q So, again, they are studying cells in a petre dish?
A Yes.
Q What does Pliskova conclude, if anything?
A Well, it is a mechanism paper. They talk about how it induces -- benzopyrene induces P53 tumor suppressor expression and abolish both S-phase arrest and apoptosis induced by the PAHs.
Potentiated deprecative effect of BaP. Thus, specific genotoxic and non-genotoxic event for interacting on the effects of BaP cell proliferation.
Q How about in layman's term, what are we looking at?
A I think the reason that I thought this was important is because of this notation about non-genotoxic mechanisms which hadn't been talked about too much on any other paper.
Q Well --

A And they also talk about the BAP and TCDD have some similar toxicities.
On Page 254, in the right-hand column, in the first full paragraph, about halfway down it says,
"Using a combination of DNA staining and detection of BrdU incorporation, we found that like TCDD, BAP and BAA also partially inhibited induction of S-phase entry by E2. However, unlike TCDD, both BaP and BaA also stimulated G1-S-phase transition, when applied to serum-starved cells, albeit to a lesser extent than E2 itself. Interestingly Dibeno[a,h]anthracene, a strong AhR ligand, which has been shown to be antiestrogenic in MCF-7 cells, had the same effect as TCDD both on the E2-treated and untreated cells. These results seem to support the hypothesis that unlike other PAH's, BaP and BaA, or their metabolites that are less efficient inducers of AhR-mediated activity, can activate ER

and stimulate cell proliferation."
Point being, that they have similar toxicity of TCDD and that the effects of the two together are going to be at least additive.
Q Similar effects, when they contact the Ah receptor; right; that is what they are saying?
A No, this is talking about other effects. That is the point I was trying to make.
Non-Ah receptor stimulated toxicity, because these other types of toxicity to the cells are not related to the age receptor. And they are pointing that out. That's all.
Q Let's look at the first page of the article. This is on the right-hand column, about an inch or so down, she says, "Today, PAHs are regarded mostly as antiestrogens principally due to their ability to activate aryl," A-R-Y-L, "hydrocarbon receptor," that is AhR receptor, "which may lead to supression of estrogen response element controlled gene expression."
So they are talking about the PAHs being protective in some measure; is that right?
A No. Stimulating Ah receptor creates adverse

effect. It influences the ability to the cell to regulate its growth properly.
Q Does an antiestrogen cancel out an estrogenic compound?
A Yes, it would.
Q What is the relevance of the Pliskova paper to Sherrie Barnes?
A Well, I have been trying to say that, to me, it addresses the issue of the dioxin, plus the PAHs being more harmful.
Q Okay. Does it identify a particular dose or exposure level in which harm would occur?
A No. It is an in vitro study. It wouldn't have any quantitative value.
Q So it is hypothesis generating in that regard?
A No, it demonstrates in an in vitro system, a mechanism. Those give us insights into why we would have this young woman developing such a malignant cancer at such a young age following in vitro, in utero, and early childhood exposure to these chemicals.
Q Well, it looked at particular PAHs; is that right?
A Yes, they looked at two particular PAHs.
Q They did not look at creosote as a mixture?
A Right. Correct.

Q Which -- which congeners of dioxin did the Pliskova paper study?
A TCDD.
Q So you had one congener of dioxin and two different congeners of PAH?
A I don't think they actually did TCDD. They just referred TCDD studies. They, themselves, just did PAH studies.
Q Okay. So the Pliskova paper does not actually study a synergistic effect between PAHs and TCDD?
A Correct.
Q It just shows that certain -- certain PAHs at certain levels can have a dioxin-like effect?
A There is some missing page here.
Q Sorry.
A 247 is missing.
Q I will have to supply that.
A 249 is missing. 251 is missing. 252 is missing.
Q You just got the even pages?
A So I am looking for things like what they used, but the pages are missing.
Q But from the abstract -- and I apologize for that, Doctor. We will supply a full copy when we come back to this in our next session.

From the abstract, it looks like they were not studying the synergistic effect; is that right?
A No, they were not studying the synergistic effect. I am simply saying that that is one the reasons why it is relevant.
Q It suggests --
A It suggested that the two together are going to have a more likelihood of developing the cancer.
Q Can we do one more paper and then call it quits?
A We will do Revich.
MR. HOPP: Keith, you all right? Can you hang in there?
MR. PRUDHOMME: Sure.
BY MR. HOPP:
Q Let's do Revich. The next document on your list, Doctor, 20, is the Revich paper; right?
A Yea.
Q Handing you what we marked as Exhibit 150.
(Defendants' Exhibits 150 was marked for identification by the court reporter.)
BY MR. HOPP:
Q This is the Revich paper; right?
A Yes, sir.
Q And Revich is looking at dioxin exposure in

Chapaevsk, C-H-A-P-A-E-V-S-K, Russia; is that right?
A Yes.
Q What happened in Chapaevsk, Russia to make Revich want to study dioxin exposure?
A There was a pesticide plant there that made chlorinated pesticides.
Q In particular, TCDD; right?
A No. Nobody makes TCDD, but they were making lindane.
Q Lindane. Okay.
A And they generated a huge pollution with TCDD, TEQs. There was a -- they had levels that are a little bit higher than the levels that we have outside the Koppers plant in Grenada, but not too much higher. There is certainly a good overlap there.
Q Okay. All right. Did Revich find an increase incidence of breast cancer in this exposed population?
A Yes, I think that is the point. The Chapaevsk -- how did you pronounce it?
Q Chapaevsk.
A Chapaevsk women had a higher risk overall due to breast cancer. 2.1, at 1.6 to 2.7 and then some other cancers, as well.
Increase female breast cancer in all age groups compared to Russia and the Sumara region in 1998. There

is a table in here. I think a graph -- a figure that -- Figure 1 and Figure 2.
Figure 2 is the female breast cancer rate. 958 is the page. And it shows consistently at all ages higher breast cancer rate for women in that region.
Q Compared to the rest of Russia and to this other area, the Sumara area?
A Yes, which is probably the general area that this thing is in. And they also have some data on concentrations of the PCDD and PCDF in the blood, milk; soil; air; and they also have some data on how far away they were from the plant for concentration of blood.
The -- I think this is TEQ -- yes. Picogram TEQ on Table 13, they had six people that they studied which was within one to three kilometers of the plant.
Their values were 75, picogram TEQ, opposed to those that were five to eight kilometers away where it was four people. And their value was 24. And then they did some other control values.
Q So is this like a cohort study or a cross-sectional?
A This is cross-sectional, environmental, biomarker and -- yeah, cross-sectional study. I don't think they had any controls. They used, as I said already, published rates.

Q So did Revich, in looking at breast cancer, attempt to control other known risk factors?
A Let me see what he did in terms of that issue. I think he assumed that -- I don't see that he did any analysis, for example, age of menarche, menopause, and all of that other stuff.
Q Right. Now, he did identify exposure levels or at least --
A Yes, he had exposure levels.
Q And did he identify the level at which the exposure is likely to cause harm?
A Well, I don't think we can say that because he doesn't have a no effect level.
In other words, he has a level of blood TEQs in six people that lives within one to three kilometers of the center. So we can say that if you are between the background level and that level, somewhere in there would be the level at which you start seeing any excesses.
Q He does not give us a bright line for excess levels of cancer?
A Well, what they say in regulatory circles is that he gave a single value that was the only and, therefore, the lowest observed adverse effect level of 75 in the blood.

Q 75 picograms per gram?
A Yes, picograms per gram.
Q And that is total TEQ?
A Total TEQ.
Q Which is much higher than the level that was measured in the cohort in Grenada; correct?
A Well, it is a -- it is higher than the average.
Q Total TEQ in Grenada was 34; right?
A Not the highest values, no. I think we had some others that were high.
Let's see if I can find where I did look at this. We have one -- I think one of these values. The TEQ was 92 on one of our folks and another one was 93. One had 50. One at 89. So we had some that were clearly up in that range.
Q Okay. But --
A Yeah, the mean value is whatever we said it was.
Q But what is the mean value in the Revich paper?
A 75. They didn't give the breakdown. Yeah, the mean value is higher. I agree with that. Now, there is also some soil values here.
Q Well, let's start with the workers -- female workers' blood. The workers had a total TEQ of 412; is that right?

A Yeah, that is the workers. I am talking about people who were living next to the site.
Q One to three kilometers, it was 75.2?
A That's right.
Q And for his analysis of breast cancer, does he combine the workers and women who lived near the plant or does he examine just the women who lived one to three kilometers from the plant?
A I think he may have combined them, but I don't know, looking at this. Yes, that was four workers who worked in the plant. And that he refers to an earlier paper that he published that report.
Q Six women who lived from one to three kilometers?
A And there was six women who lived between one to three kilometers. That is where the 75 came from. That's also from an earlier paper.
Q So workers from one to three kilometers combined, that is a total of ten women; right?
A Yes. Well, it does not say that they are all women; do they?
Q Yeah, look at Table 4. Female blood?
A One to three kilometers. Four -- okay. The 75 is the one -- is the six there?
Q Maybe it is a bit obscure, I mean, the title of

the table says Female Workers Blood and then the column, one to three kilometers, that is not workers. So it is a bit ambiguous; isn't it, with respect --
A Yeah, it is. That is an interesting question. Are they all women or is this men and women? Let's see, we are talking about dioxin and public health. The guy is not a really skilled writer.
Q Well, he is Russian.
A Well, it is not his native language. It's hard for them to sometimes get it straight. Even I have had Russian papers that I read and had them translated, and they were really awful.
But here is an example of some complexity that is hard -- blood samples were taken from 14 people. 90 percent of women lived in Chapaevsk versus for more than three years. So maybe it is all women.
Q Total on Table 4 is 14. You got four workers, six --
A Yeah. 90 percent of the women -- it must be all women.
Q Okay.
A But it does not say that anywhere.
Q All right. In any event -- at any rate, Revich identifies the effect level being 75 picograms per --
A Yeah. Yeah. We do have something to look at.

MR. HOPP: Okay. All right. Shall we knock off for the day? It is 5:00 o'clock.

THE WITNESS: You won't get an argument out of me.

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I, the undersigned, a Certified Shorthand Reporter of the State of California, do hereby certify:

That the foregoing proceedings were taken before me at the time and place herein set forth; that any witnesses in the foregoing proceedings, prior to testifying, were placed under oath; that a verbatim record of the proceedings was made by me using machine shorthand which was thereafter transcribed under my direction; further, that the foregoing is an accurate transcription thereof.

I further certify that I am neither financially interested in the action nor a relative or employee of any attorney of any of the parties.

IN WITNESS WHEREOF, I have this date subscribed my name.

Dated: _____

_____

Diana Janniere
CSR No. 10034

I, JAMES DAHLGREN, M.D., do hereby declare under penalty of perjury that I have read the foregoing transcript; that I have made any corrections as appear noted, in ink, initialed by me, or attached hereto; that my testimony as contained herein, as corrected, is true and correct.

EXECUTED this _____ day of _____, 20__, at _____, _____.
(City) (State)

_____

JAMES DAHLGREN, M.D.

E